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TUBERCULAR MENINGITIS; WITH REPORT OF A PECULIAR CASE.

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Among the diseases that are generally regarded as affections of early life, there is not one that is really so little understood and so unsuccessfully treated as tubercular meningitis. While this disease is not an uncommon affection, it is very often the case that a post-mortem reveals the fact that a wrong diagnosis had been made which is only proven when it is too late. This, then, brings up the question regarding a primary and secondary form of this disease and it may well be asked—"Why does tuberculosis first make its appearance in the form of meningitis, and why does it not show symptoms of a general tuberculosis before becoming localized in the meninges?"

If the authorities of most of our writers can be recognized wherein they assert that this disease is almost always secondary to the affection in some other part of the body, why do we find tubercular meningitis in children who have been apparently healthy prior to the attack of the disease?

Many eminent writers assert that in primary cases, which are especially frequent in children and young people, it seems to arise in persons previously quite healthy, or at most after a few weeks' malaise; but even in these instances, after death, *it is nearly always the case that some other lesion is present*, such as caseating bronchial glands, or miliary tuberculosis of the lungs and other viscera, or a caseous nodule in the brain itself.

Generally speaking, tuberculosis invades the body or it becomes susceptible through three and possibly four channels: hereditary taint, partaking of milk from a cow infected with tuberculosis, nursing by a tubercular woman, and possibly by the bacilli finding an entrance through a wound. It is only in rare cases, however, that it invades the body through the last-mentioned channel. As regards a hereditary

predisposition there is no doubt; as may be said, also, of the infection through cow's milk and the breast milk of the mother or wet nurse, infected with tuberculosis.

I have referred to this disease as being little understood and unsuccessfully treated, by which I wish to convey the idea that while we know of the bacilli of tuberculosis, know the results of its invasion of the body and the very unsuccessful treatment of the conditions that arise, we are very much in the dark regarding a diagnosis and causes that localize this disease without first showing symptoms of a general or miliary form which reasonably should occur.

It is not difficult to find a cause for tubercular meningitis when the patient is already affected with phthisis, hip-joint disease, caries of the spine or other tubercular or strumous complaints, which make the disease secondary; but the reason for a primary form of this disease is very much in the dark, and our knowledge of the local invasion, especially in the form of meningitis, is very little.

Etiology.—This disease occurs at all ages, but is generally regarded as more frequent in children than adults, and it is very certain it affects males more than females. It is claimed by medical writers in general that so far as its causation is concerned, it is constantly associated with tubercle elsewhere in the body, and is really secondary. Primary cases which are found in children and young people, seem to arise in persons previously quite healthy or after a few weeks of ill health; still it is claimed that even in such cases the condition is due to tuberculosis in some other region of the body. There may be a discharge from the ear, but if it has any relation to the disease, it is either that it indicates general ill-health, or that it opens a passage for the entrance of the tubercle bacilli. Sometimes the glands of the neck enlarge, break down and suppurate prior to this disease being manifested.

We know that the cause of tuberculosis, no matter what part of the body is affected, is due to a bacillus, which may invade the body through infected cow's milk or the breast milk of a tubercular woman and possibly through a wound. Experiment has clearly shown that the introduction of tubercle bacilli into the tissues will produce tubercles; and this must be by some special irritant properties of the bacillus. In relation to the occurrence of tuberculous disease in man, we have before us the question, how is it usually introduced into the system, so as to produce the numerous tubercular lesions of the bones, joints, lungs, peritoneum and other organs? If this cannot in every case be answered, it is in many instances suf-

ficiently obvious. The bacillus may enter from without through the mucous passages, of which the respiratory gives the preponderating number of instances; thus, tuberculosis of the lungs follows the lodgment of the bacillus in the bronchioles or lung tissue. With comparative rarity, the bacillus may enter through a wound. In most cases we must suppose a special predisposition on the part of the individual (hereditary), or of the tissue first affected (depressed vitality from inflammation), which allows the tubercle to establish itself and thrive. Allowing this to be true, it still does not account for a primary meningitis being manifested when it is so reasonable to suppose that other organs of the body, more exposed, should be affected first and show unmistakable symptoms of the disease.

Morbid Anatomy.—The characteristic appearances are seen in the pia mater and consist of the effusion of lymph, and the presence of tubercles. The lymph is gelatinous and translucent, or more opaque, and gray or a grayish-yellow, rarely or never purulent and is contained in the meshes of the pia mater, especially at the base of the brain, over the chiasma, the space behind it and the adjacent crura and pons. It commonly extends into the Sylvian fissure on each side, along the course of the middle cerebral artery. The surface of the hemispheres is free from lymph, but it is found at the top of the cerebellum, at the anterior part. With the lymph are mixed tubercles, of different sizes, gray and opaque, occasionally commencing to caseate. Under the microscope the smaller tubercles present lymphoid corpuscles in the perivascular sheath; the larger tubercles may present giant cells and bacilli. There may be abundant lymph in the characteristic situations, with few, if any, tubercles, or there may be a number of tubercles with little lymph. Occasionally there may be symptoms indistinguishable from those of tubercular meningitis, in which tubercles are found on the surface with no meningitis. The ventricles of the brain are commonly distended with fluid, the convolutions are flattened against the skull, the fornix and septum lucidum are generally soft, and the ependyma of the ventricles presents a granular or sanded appearance. The cranial dura mater is not usually affected, but the spinal dura mater sometimes shows minute tubercles, and lymph may extend from the pia mater to the cervical region of the spinal cord. A general tuberculosis is not uncommon. Tubercles may be found in the lungs, liver, spleen and kidneys. In both secondary and primary cases the modern view is that the meninges are infected from a preceding tubercular

deposit in the lung, kidneys, bronchial gland, brain or elsewhere; or possibly from outside.

Symptomatology.—The symptoms will be first described as they commonly occur in children, and the differences in secondary cases afterwards mentioned.

There is often a stage of ill health. The child is restless, loses appetite, may be occasionally sick and has constipation. The illness begins more definitely by headache, vomiting or a convulsion. The headache is severe and continuous, and there is moaning or occasionally a sudden cry. There is a moderate degree of fever, quick pulse, sensibility to light and sound. The child shuts its eyes and resents being disturbed. The vomiting does not generally last long, and the convulsions that occur at the beginning are seldom repeated.

After a few days there may be slight delirium and the patient becomes drowsy. The head is sometimes retracted and neck stiff. The abdomen becomes hollowed out or retracted, the margins of the ribs and iliac crests being prominent. The pulse may be slow, and often irregular; the respirations slow, sighing and irregular; the temperature is generally high, oscillates between 101° and 103°. There is a tendency to vasomotor paralysis as seen in the flushing of the face. If the finger is drawn across the skin of the forehead or abdomen, a broad red line quickly appears which may last as long as five minutes. This condition, which is not peculiar to, but only more marked in meningitis, is called the cerebral streak. Changes often occur very early in the optic disk, which at first becomes vascular and then shows definite optic neuritis. Food is taken badly and the bowels are constipated.

From this point the case may go to a fatal termination without other symptoms. The drowsiness increases to coma, optic neuritis is more marked, the abdomen becomes more hollowed, pulse more irregular, feebler and generally quicker, the respiration may take on the character of Cheyne-Stokes breathing, the temperature may fall more or less rapidly, or before death rise quickly to 106° or 107°. Mucous accumulates in the bronchial tubes and with failing pulse death takes place. It is very often the case that the last few days are marked by local symptoms. An arm or leg, or the arm and leg on one side become rigid or paralyzed. There may be facial paralysis or ptosis. The pupils are very often unequal and may be insensitive to light. In this stage convulsions may occur. With these symptoms coma becomes more profound and death takes place as above shown or the patient is asphyxiated in a convulsion.

The duration of the illness varies between ten days and three weeks from the beginning of the pronounced symptoms, but it may be five or six weeks. The above course of the disease has been divided into three stages—irritation, compression and paralysis, but it is not always easy to distinguish between them, and in some cases the more typical symptoms may be little marked, coma alone being prominent.

In secondary tubercular meningitis the symptoms are often more rapidly developed. The patient may, with little warning, become delirious or have paralysis of a limb or face, or have a convulsion, quickly becoming comatose, and dying in a few days.

Diagnosis.—This is sometimes easy, but at other times difficult and impossible until late in the illness. We must expect meningitis when there are decided head symptoms accompanied by fever, but headache alone would not be a diagnostic symptom. In young children otitis may cause headache, moaning, vomiting, photophobia and the desire to be undisturbed. A careful examination of the ear and mastoid process may reveal this localized condition. Enteric fever may for some days resemble meningitis, but in that fever, headache rarely persists after the tenth day, and generally by that time the bowel movements or the rose spots on a full abdomen, will decide the diagnosis, which will be confirmed later by the absence of convulsions, rigidity or paralysis. The mistake is often made in cases of meningitis, without prominent headache, but with flushed face, delirium and pyrexia, they being diagnosed as enteric fever. The most useful symptoms are irregular pulse, sighing or irregular respiration, rigidity of muscle or paralysis, convulsions and optic neuritis. However, optic neuritis may occur in enteric fever. Tubercle of the choroid only occurs in a small percentage of cases and is not to be depended upon alone for a diagnosis. In young children, decided cerebral symptoms accompany other acute sickness, as in pneumonia and broncho-pneumonia; there may occur drowsiness with a retracted head, and convulsions may occur towards the end. These symptoms would be explained by the detection of localized dullness with bronchial breathing, but râles over the whole chest might indicate a general tuberculosis. The exhaustion following mal-nutrition, bad feeding or severe diarrhœa in young infants may simulate this disease. The child is drowsy or comatose, with pale face, sunken eyes, dilated, irregular pupils, and irregular, sighing respiration. It was formerly called hydrocephaloid disease. It is distinguished from meningitis by the history, absence of

fever and local paralysis, the depressed fontanelle and its speedy improvement under supporting treatment. The distinction of the tubercular from other forms of meningitis rests largely upon the absence of local cause for a suppurative meningitis, as cranial injury or otitis; the previous existence of strumous or tubercular lesions, the paralysis of cranial nerves, indicating that the meningitis is situated at the base rather than over the vertex; the duration, which is generally much shorter in suppurative meningitis. So far as the clinical history goes, and the absence of any local exciting cause, the same diagnostic points cover those cases of meningitis which mostly affect infants or very young children, in which no tubercles can be found, but only a grayish lymph at the base. In every respect but the absence of tubercles they resemble tubercular meningitis, from which they cannot be distinguished during life. With meningeal symptoms and tuberculosis of the choroid a diagnosis may be made of tubercular meningitis. With meningitis a lumbar puncture may give a diagnosis also.

Prognosis.—The prognosis in this disease is very grave, and it is a question whether there is ever a recovery. In the cases reported as recovering, there is a just suspicion that a wrong diagnosis had been made. We rarely find traces of a past tubercular meningitis in those who die of other diseases, and it is very difficult to prove during life that the case is tubercular, even if meningitis is present. Still a certain number of patients with apparent tubercular meningitis do get well. The recovery is slow, the speech, vision, etc., remaining imperfect for weeks or months. The prognosis, then, is unfavorable, especially in the secondary form where phthisis, hip disease or other well-marked tubercular lesions are present. Yet primary cases need not be considered absolutely hopeless.

Treatment.—With the prognosis unfavorable, the treatment is reduced to a small limit. Cold should be applied to the head by means of an ice bag, the bowels kept open and milk given in small quantities, frequently. Blisters are sometimes applied to the back of the neck, but are of doubtful value in the tubercular form of meningitis. Irritant applications have been used upon the close-shaven scalp, but here, too, they seem to be generally of little benefit. In some cases iodoform ointment may show some beneficial results. Of the internal remedies, iodide of potassium has been extensively used in the hope of influencing the morbid process. The bromide is given to allay the pain in the head. A combination of the five bromides has also been used.

A Somewhat Peculiar Case.—Knowing that cases of this disease are always interesting to the profession, and especially so when they vary from the usual course, I will give a history of one that I recently discovered which assumed a somewhat different form from the typical ones. The history of the case with its developments made the diagnosis reasonably sure, although no post-mortem was performed. The peculiarity of the case is in its changeable nature, for although the child was paralyzed from time to time, this would pass away, then occur again. The child had several spells of apparent collapse but would recover. As early as the fourth day the arm and limb on the right side were paralyzed, which passed away after a few days. Then the left arm and limb were affected, which also passed away, and finally, when the child died on the seventeenth (17th) day, there were no signs of paralysis. Another somewhat uncommon condition was the fact of the temperature gradually rising several days before death, instead of the sudden rise that occurs so often. The case is as follows:

A few weeks ago I was called to see a little girl one year of age who had been slightly ill for about a week previous. There had been no marked symptoms until the day I saw her when she was taken with vomiting and convulsions. I had treated the child a short time before for enlargement of the glands of the neck and a discharge from the ear, from which she fully recovered. The mother informed me that for the past few weeks the child had shown symptoms of her milk disagreeing with her, and had nursed but very little. Plenty of cow's milk had been given and it had received sufficient nourishment. While treating the child for its former trouble I got a history of tuberculosis, the mother's two sisters and her mother having died from it in a pulmonary form. I advised the mother to wean the child at once on account of the possible danger of its contracting the disease, the mother being predisposed, although no signs of tuberculosis were manifested as yet. The mother, however, did not heed my warning and continued to nurse the child and also gave it cow's milk.

At my first call I found the child in convulsions, pulse 110, temperature 100° (axilla), respiration about normal. There had been several convulsions and vomiting and the child nursed badly. On the second day the pulse reached 120, temperature (axilla) 101°, respiration irregular and quickened, with no convulsions or vomiting, which never occurred again. The pupils were dilated and unequal, which continued throughout the course of the disease. The tem-

perature then indicated tuberculosis inasmuch as it had the evening rise and the morning fall. On the third day the pulse reached 136, temperature 101° (axilla) and Cheyne-Stokes breathing. Fourth day symptoms about the same with paralysis of right arm and limb lasting about forty-eight hours; coma. From the fourth to eighth day symptoms about the same, temperature varying from 101° to 103° (axilla). On the eighth day the arm and limb on left side became paralyzed. From the eighth to eleventh day, symptoms the same with a gradual elevation of temperature. Eleventh day, pulse irregular and hard to count, temperature 104° (axilla), other symptoms the same. Twelfth day more normal pulse and respiration, temperature remaining at 104° (axilla). Thirteenth day, temperature 102½° (axilla), collection of mucus in bronchial tubes, other symptoms the same. Fourteenth day showed pulse 140, respiration oppressed, temperature 104½° (axilla), with increased collection in throat. Fifteenth day no change. There were no symptoms of paralysis. Sixteenth day, throat paralyzed. Up to this time nourishment had been given by the mouth, although coma was present. Nourishment was now given by the rectum. The collection of mucus increased, respiration oppressed, heart weak, temperature 104° (axilla), child in a state of collapse. Seventeenth day, child died from failure of heart. There were no symptoms of general paralysis. Unequal dilated pupils present. The other symptoms in this case did not differ from a typical case of the disease. The bowels were constipated, abdomen sunken, cerebral streak, delirium, dilated unequal pupils, etc. I examined the child both at morning and night for several days and noted the evening rise and the morning fall of the temperature. A careful examination of the case showed no symptoms of any other disorder, not even a general or miliary tuberculosis, and a cause for the condition in this case might be due to a hereditary taint as the child was strumous, from being fed on cow's milk infected with the germ or *possibly* by the entrance of the infection through the discharging ear. The mother's milk was not analyzed as she showed no signs of tuberculosis, although there is no doubt a hereditary predisposition.

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